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IMMUNE RESPONSES IN PARASITIC DISEASES.(U)
JUL 76 D J STECHSCHULTE, H B LINDSLEY

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Report Number 1 ✓

"Immune Responses in Parasitic Diseases"

Annual Summary Report

Daniel J. Stechschulte, M.D.
Herbert B. Lindsley, M.D.

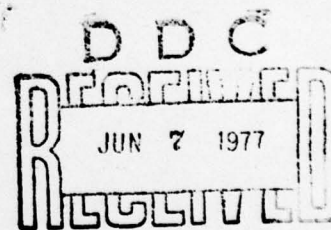
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1. The effect of Trypanosoma rhodesiense 1886 infection in the rat results in significant stimulation of the IgG1 and IgM immunoglobulin levels. It was not possible to determine whether this was antibody for one or more of the trypanosomal antigens or if it was nonspecific stimulation of immunoglobulin production. The effect of this parasitic infection on an ongoing antibody response to the hapten DNP was evaluated by immunizing rats with DNP-BGG followed by an infection with T. rhodesiense at a time when antibody production with hapten specificity is ongoing. Potentiation of the IgG1 antibody response to DNP was observed but no potentiation of the IgM response was noted. This would suggest that programmed IgM antibody production was unaffected by the parasitic infection whereas an ongoing antibody response in IgG1 class was modulated by the infection. The presence of a low molecular weight and a 19S species of IgM immunoglobulin was demonstrated subsequent to infection with T. rhodesiense but it was not possible to demonstrate antibody specificity in these molecular species of IgM.

In a related study

2. T. rhodesiense 1886 organisms were purified by DEAE-cellulose chromatography, sonicated and the insoluble debris separated by high speed ultracentrifugation. The soluble proteins were further fractionated by DEAE-cellulose chromatography, gel-filtration chromatography and assessed by analytic isoelectric focusing. The initial approach was to radiolabel the soluble proteins so that a radioimmunoassay for the presence of antibody could be established. Only moderate success was achieved in labeling trypanosomal materials. Additional techniques for obtaining radiolabeled antigen are currently in progress.

3. In a collaborative study with Dr. Ray Naegle it was demonstrated that rats infected with T. rhodesiense 1886 developed evidence of glomerulonephritis. This is associated with a depressed complement level presumably due to consumption of complement proteins associated with circulating immune complexes. Molecular species of immune complex, size and preliminary quantitation in circulation are studies that are in progress. These studies suggest that even though T. rhodesiense infection in the rat tends to be a fulminate infection there is presence of a glomerulonephritis at 4 weeks following infection.

4. Utilizing monospecific anti-IgG1 it has been possible to isolate hapten specific gamma 1 antibody from rat serum. The gamma 1 immunoglobulin level varies from 1 to as high as 7 milligrams per ml concentration in rat serum. In view of the fact that early studies demonstrated a significant increase in this immunoglobulin level during T. rhodesiense infection in the rat this immunoglobulin class will be extracted from infected rat serum utilizing antibody specific affinity chromatography. The gamma 1 immunoglobulin that can be eluted from these columns and recovered in milligram amounts will constitute the first attempt at demonstration of protective antibody in this parasitic infection.

5. To date no progress has been made in assessing the role of lymphocytes by adoptive transfer in the host-immune response to this infection.

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